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# THE EFFECTS OF OVERNIGHT SLEEP DEPRIVATION OR TOTAL SLEEP DEPRIVATION ON THE BODY

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## LITERATURE REVIEW

**Abstract**

This analytical and scientific work explores the multifaceted effects of overnight and total sleep deprivation on the human body. Sleep deprivation, increasingly prevalent due to the modern pace of life, significantly impacts various physiological systems including the cardiovascular, nervous, reproductive, digestive, and immune systems. It also leads to hormonal imbalances and cognitive impairments, manifesting in serious somatic and mental illnesses. Despite extensive research, the mechanisms through which sleep influences these body systems remain incompletely understood. This review aims to synthesize current knowledge on the subject, emphasizing the importance of early identification and treatment of sleep disorders to prevent a range of health issues and enhance overall well-being.

**Keywords**: sleep deprivation, sleep disorders, neurophysiology, insomnia, stress, cortisol, neurotransmitters, melatonin, health effects, mental health, cardiovascular desease

Introduction. Despite sleep being recognized as a fundamental element of health, economic prosperity, and social functioning, the modern pace of life often compels individuals to overlook its significance. Sleep duration varies significantly throughout life and shows an inverse correlation with age. However, the World Health Organisation (WHO) recommends that adults get at least 7-9 hours of sleep per night. Nevertheless, statistical surveys indicate that at least one third of the world's population is prone to sleep deprivation, 10% (typically in industrialised countries) suffer from chronic sleep disorders, and approximately 5-7% suffer from diseases caused by regular sleep deprivation. Moreover, individual bodily requirements cannot be overlooked, as some people can live effectively with just 6 hours of sleep, while others feel exhausted even after 10 hours of sleep

Total or chronic sleep deprivation can lead to serious somatic and mental illnesses of varying complexity. For example, inadequate nightly rest affects the cardiovascular, nervous, reproductive, digestive and immune systems, induces hormonal imbalances and can trigger the onset of various cognitive impairments, mental disorders and even depression and suicidal thoughts. Unfortunately, the mechanisms of sleep's influence on various body systems are still not fully understood and require careful experimental study and statistical analysis. Early identification of the cause and development of proper treatment for sleep disorders will contribute to the prevention of numerous diseases and dysfunctions in the body. This will also have a pronounced positive economic effect, as a mentally healthy and energetic person is more competitive and efficient.

Mechanisms of occurrence and characteristics of the sleep process

Sleep is a complex physiological process regulated systematically, regionally, and locally by both cellular and molecular mechanisms. It is clear that sleep is necessary for many vital functions, including development, maintenance and regulation of energy expenditure, modulation of immune responses, cognition, productivity, alertness, psychological well-being and many others. Overall, the definition of sleep in mammals depends on the metabolism and activity manifested by electrical signals in the brain as recorded by an electroencephalogram (EEG).

When a person sleeps, it may appear that they are unconscious with closed eyes. However, the cortex of the brain remains active, and the registration of brain activity has allowed the identification of waves that characterize brain function. These sleep phases consist of two main types: rapid eye movement (REM) and non-REM. The non-rapid eye movement phase has three stages (N1, N2, and N3), characterized by increasing sleep depth, slower EEG waves, longer duration, followed by rapid eye movement sleep (REM). Throughout the night, human sleep alternates between NREMS and REMS in cycles occurring approximately four to five times lasting 90 minutes each [1]. Sleep is typically deeper at the beginning of the cycle, with the REM period occupying more of the cycle as the process continues to lengthen.

Ekonomo identified areas of the brain that modulate sleep by studying people who contracted "encephalitis lethargica" during the global influenza epidemic of the late 1920s. He concluded that the brain region responsible for inducing this process is located

in the anterior hypothalamus, while the region promoting wakefulness is located in the posterior hypothalamus [2]. Further research has identified distinct channels (networks, pathways) that interconnect arousal, non-rapid eye movement sleep (NREMS), and rapid eye movement sleep (REMS). These networks regulate sleep and wakefulness through projections of neurons to local and distant brain regions, and substances influencing the process can be produced by the surrounding cells themselves and cells in distant brain regions [3].

There is a certain balance between molecules associated with wakefulness and molecules that promote sleep, and this balance can be modulated by brain regions that dominate in the production of certain molecules, thereby influencing states of wakefulness. Sleep-promoting substances include adenosine, extracellular ATP, prostaglandins, anti-inflammatory cytokines and GABA. "Arousal-promoting substances include glutamate, acetylcholine, norepinephrine and hypocretin [4]. Despite all the progress made in understanding the mechanisms of sleep over the past decades, it is worth noting that this process is regulated by pathways and substances that also perform other physiological functions, complicating our understanding of the functions of sleep and sleep-related pathologies.

It's also worth noting that neurotransmitters such as acetylcholine, dopamine, norepinephrine, histamine, serotonin, hypocretins (also known as orexins), neuropeptide S and glutamate typically promote wakefulness or alertness. For example, hypocretins in the hypothalamus are considered to be among the most important regulators of sleep because they stimulate and maintain wakefulness by promoting the release of arousal-inducing neurotransmitters (such as norepinephrine, dopamine, acetylcholine and histamine). This is achieved by their ability to activate specific neurons that release the aforementioned neurotransmitters [5]. As for the function of GABA, which is found in most neurons, it largely inhibits the activity of glutamatergic neurons and their respective receptors, thereby promoting NREMS. It acts as an inhibitory neurotransmitter through α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) or N-methyl-D-aspartate (NMDA) receptors, primarily promoting inhibition and suppressing sleep [6]. Antagonists of GABA include benzodiazepines, barbiturates, imidazopyridines and cyclopyrrolones, which inhibit excitation and induce drowsiness; despite their low levels, their interaction with the receptors is strong.

In terms of molecular mechanisms, substances directly involved in the regulation of energy release and storage (extracellular ATP, adenosine), which subsequently interact with purine, as well as those shared with certain pathogens (e.g. bacteria), perceived by specific receptors, are amplified by certain brain activities during wakefulness, signalling sleep-regulating molecules (interleukin 1 beta (IL-1B), tumour necrosis factor (TNF-α)) and thus inducing sleep. Subsequently, sleep-regulating molecules signal neurotransmitters (N-methyl-D-aspartic acid, NK-1, IL-1R), which in turn modify ion channels ($\boldsymbol{Ca^{2+}}$and $\boldsymbol{K^{+}}$), causing changes in excitatory and inhibitory postsynaptic potentials and changes in sleep phase and EEG [7].

It is worth mentioning another very important regulator of the sleep process - melatonin. This is a methoxyindole synthesised and secreted mainly by the pineal gland at night under normal light/dark conditions. The endogenous secretion rhythm is generated by the suprachiasmatic nuclei and synchronised with the light/dark cycle. Light can either suppress melatonin production or synchronise it according to the light schedule. Melatonin secretion typically begins around 14 hours after spontaneous awakening (which corresponds to 21:00 in a sighted person who wakes up at 07:00) [8]. Melatonin typically begins to exert its effects approximately 2 hours after the onset of secretion, and depending on the concentration, the potency of the hormone's sleep-inducing effect is similar to that of the powerful sleep aid zopiclone [9]. Thus, during sleep preparation, melatonin induces heat loss, reduces arousal and associated brain activation, and suppresses cortisol production.

The stress hormone cortisol is the end product of the hypothalamic-pituitary-adrenal (HPA) axis, and the cortisol awakening response (CAR) is a rapid increase in cortisol levels observed immediately after waking. Thus, during the waking period following a night's sleep, cortisol levels typically increase by 38%-75% (due to the CAR), peaking approximately 30 minutes after the last wake-up. Numerous studies suggest that the function of this hormone may be associated with general alertness, increased energy and improved mood, so that a lack of cortisol can lead to weakness, apathy and drowsiness [10].

Causes of sleep deprivation. Sleep disturbance is a complex phenomenon and its occurrence can be influenced by a number of factors (apart from diseases such as multiple sclerosis, various mental and neurological disorders, etc., which cause sleep disturbance as a symptom) that interact with each other. According to research in the fields of somnology and neurobiology, there are several key factors that can directly influence the quality and duration of sleep. Here are just a few of them.

Stress has been shown to be a significant cause of sleep disturbance and the onset of insomnia. Many situational factors strongly influence sleep after stress, such as whether the stressful factor was controlled and anticipated or predictable, whether the individual had the opportunity to adapt to the situation, and the relative individual resilience and vulnerability of the person experiencing stress. Many brain regions and neurochemical systems have been identified that interact to create the link between stress and sleep. The neurochemical components include various monoergic neurotransmitters, corticotropin-releasing factor, hypocretins and prolactin, and the brain regions include the hypothalamus, amygdala and prefrontal cortex. These areas are also thought to play an important role in memory processes and in how stressful memories can affect arousal during sleep, sometimes even causing unpleasant dreams[11].

Caffeine is the most widely consumed psychoactive substance in the world. It is readily available in coffee, energy drinks, some medications, and other products and beverages, and is typically used to reduce sleepiness, increase alertness, and increase general alertness in the body. Numerous experimental studies show that this substance shortens the period of non-rapid eye movement sleep (NREMS) and, conversely, prolongs rapid eye movement sleep (REMS), promotes an increase in cortisol levels, thereby significantly reducing overall sleep duration and impairing sleep quality. Frequent caffeine overdose leads to persistent disruption of circadian rhythms (including the 'sleep clock'), often causing chronic insomnia and increasing the risk of neurological and mental disorders.

The link between artificial light and sleep disorders is quite clear. People who work shifts are most affected, as well as those who work with any device that emits light or single very bright flashes, which actually cause a disruption in melatonin release. These people tend to fall asleep very late at night and have difficulty waking up in time for work, school or social commitments. There are also many studies on the negative impact on sleep of the so-called blue light emitted by various devices (smartphones, computers, televisions...)[12, 13].

Irregular sleep patterns and inconsistent daily activity schedules can disrupt the body's natural biological rhythms, which can affect the quality and duration of sleep. In addition, intense physical activity before bedtime can stimulate the nervous system and make it harder to fall asleep. Poor eating habits, especially in the evening, can contribute to gastrointestinal disorders and disrupt the normal course of sleep.

Disorders due to lack of sleep. The impact of sleep deprivation on cognitive function. The effects of sleep deprivation on neurocognitive processes have been well established for some time. Chronic sleep restriction is likely to induce long-term neuromodulatory changes in brain physiology, which may explain why recovery from it may take longer than from a single acute episode of sleep deprivation. In addition, numerous neuroimaging studies have shown that the hippocampus (responsible for memory consolidation) and the prefrontal cortex of the brain (whose main function is to coordinate thoughts and actions according to internal goals) are particularly sensitive to chronic sleep deprivation [14]. However, even one night of total sleep deprivation significantly reduces performance on neuropsychological tasks such as working and short-term memory assessment, decreases attentional focus, decision-making speed, task and attention switching, induces emotional fluctuations, and leads to an overall significant slowing of thought processes.

It has been shown that sleep deprivation of less than 6 hours over three to four days can slow reaction times to external stimuli (such as loud noise, bright flashes of light, rapid movement, etc.) by approximately 35% [15]. This poses a significant risk to road users and impairs the ability to make quick decisions in life-threatening situations.

It is known that the human brain consists of approximately 86 billion neurons, and each neuron can connect with thousands of other neurons, resulting in approximately 100 trillion neural connections, or synapses. Synapses are the connections through which information is transmitted from presynaptic to postsynaptic neurons, typically via neurotransmitters released from presynaptic axon terminals, which then bind to receptors on postsynaptic dendritic effectors [16]. Chronic sleep deprivation or complete sleep deprivation significantly affects the speed and strength of synaptic transmission in the brain, which is one of the reasons for the negative impact of sleep deprivation on memory, cognitive processing speed, and information retention [17].

Memory is subject to dynamic changes, sometimes leading to the formation of false memories due to biased consolidation processes or the search for the "right moment" in memory. Sleep is known to promote memory consolidation through active reorganization of concepts, whereas acute sleep deprivation suppresses the functions of this so-called search. The current model suggests that the positive effect of sleep on memory consolidation is based on a covert reactivation of recently encoded memory traces, leading to a redistribution of all visual and auditory memories to other brain regions, along with their integration into existing knowledge networks, so that sleep deprivation can cause uncertainty in one's memories or even completely alter them, making them false [18].

The impact of sleep deprivation on emotional state and behaviour. It is well known that insufficient sleep or total sleep deprivation are factors contributing to constant fatigue, apathy, anxious thoughts, general nervousness and, consequently, emotional instability and sometimes unethical or aggressive behaviour. There is evidence of an age-related trend in the development of anxiety and other disorders resulting from sleep deprivation. For example, young people are 25% more likely to experience emotional dysregulation and feelings of fatigue-induced apathy than people over the age of 45. However, it has been found that the latter group generally takes much longer to return to a normal emotional state, and overall requires more time to fully recover mental health for certain disorders [19].

People who suffer from sleep deprivation and persistent sleep problems are more prone to mental disorders such as generalised anxiety disorder and bipolar disorder, more likely to have suicidal thoughts, and particularly vulnerable to depression. Depressive disorders in people with severe sleep problems occur in about 16% of cases and are among the most commonly diagnosed mental disorders [20]. It was once thought that changes in sleep neurophysiology were typical of patients already diagnosed with depression, and that disruption of the 'sleep clock' was often a primary complaint in depression. However, many studies now suggest that disturbances in sleep duration and quality are likely to be a cause, or at least a predictor, of depression.

The process of depression resulting from sleep deprivation also has molecular explanations. It has been shown that sleep deprivation contributes to increased levels of pro-inflammatory cytokines (such as interleukin IL-6 and TNF) throughout the day, leading to an overall increase in cellular inflammation. There is a direct link between depression and so-called "cellular inflammation", as inflammatory markers are higher in patients with depression than in those without, and the incidence of comorbid depression is higher in patients with inflammatory disorders [21]. In addition, counteracting endogenous inflammation is an effective means of reducing depressive symptoms, which is why inhibitors of cellular inflammation are actively used in antidepressants [22].

It's also worth noting that people who are prone to chronic sleep deprivation or frequent total sleep deprivation are more likely to experience disorders such as sleep paralysis, narcolepsy, restless leg syndrome, hallucinations and nightmares. These disorders are often associated with feelings of constant fear, anxiety and aggression, accompanied by intrusive thoughts and a sharp decrease in productivity throughout the day [23]. Numerous studies in the United States, Australia, India and other countries have found that students with poor sleep quality receive lower grades in exams and show more depression, aggression, anxiety and apathy than their peers [24].

Effect on the cardiovascular system. Data from numerous meta-analyses of experimental studies indicate that chronic sleep deprivation significantly increases the risk of developing cardiovascular diseases such as hypertension (less commonly hypotension), tachycardia, arrhythmia, and others. These conditions, in turn, can lead to blockages in various areas of the heart, cause structural abnormalities in the myocardium, and greatly increase the likelihood of myocardial infarction.

During normal sleep, there is a decrease in blood pressure compared to waking. This decrease is known as the "nocturnal fall" and is partly due to a decrease in the tone of the sympathetic nervous system. Conventionally, a 10-20% decrease in average nighttime blood pressure (both systolic and diastolic) compared to average daytime blood pressure is considered normal. Conversely, the absence of a nocturnal drop in blood pressure is considered pathological or a harbinger of disease. Short sleep duration can disrupt this cycle, resulting in both daytime and nighttime hypertension. Numerous meta-analyses of different age groups have shown a significant, consistent association between short sleep duration (< 5 hours per night) and the risk of this pathology. It has been shown that the risk of developing hypertension increases by about 21% in people with short sleep. It has also been shown that this effect is best observed in early childhood and adolescence. [25].

It has been found that people who sleep less than 5-6 hours a night (age group 20-35 years) have a 48% higher risk of ever having an ischaemic stroke compared to those who sleep 6-8 hours a night [26]. In addition, the risk of ischaemic heart disease is significantly increased in the presence of certain chronic diseases or conditions that lead to poor sleep quality [27]. In addition, people who are chronically sleep-deprived are about 15% more likely to have a stroke (both fatal and non-fatal) [28].

Impact on metabolism. Hormonal dysregulation. Sleep deprivation and general circadian disruption can contribute to weight gain, obesity and type 2 diabetes by impairing glucose tolerance and insulin sensitivity. They can also potentially alter the timing and amount of food intake, disrupting energy balance and sometimes even causing inflammation of the lining of the digestive organs. Recent experimental studies suggest that sleep restriction may increase food intake without a corresponding increase in energy expenditure.

It has been shown that during normal sleep, the total metabolic rate decreases by about 15% and reaches its minimum in the morning before waking up, in terms of the average person's circadian rhythm [29]. It is also worth noting that growth hormone and cortisol are the "night" hormones that influence the body's control of glucose levels. Growth hormone is typically elevated at the onset of sleep, with its highest levels during slow-wave sleep, while cortisol levels increase significantly in the second half of the night, especially during rapid eye movement (REM) sleep and especially before waking up. Therefore, glucose utilisation in healthy people is highest during wakefulness, moderate during slow-wave sleep and lowest during REM sleep.

Studies have shown that 4 hours of sleep over 6 nights can reduce overall glucose tolerance by 40% and the insulin-dependent glucose utilisation response by 30%, resulting in a significant and persistent increase in blood glucose levels, with subsequent deposition in certain internal organs due to the body's inability to break down the excess [30].

From the above information, it can be concluded that reduced sleep duration or various sleep disturbances are directly associated with certain predictors of type 2 diabetes, such as glucose intolerance, insulin resistance, reduced insulin response to glucose and, ultimately, a decrease in the so-called overall diabetes susceptibility index. For example, according to various data, regular sleep of less than 6 hours per night increases the risk of developing type 2 diabetes by 20-35%, depending on body mass, age, other chronic diseases and general physical condition [31, 32].

Experimental studies have shown that chronic sleep deprivation (typically ≤4.5-5 hours) and total sleep deprivation can disrupt the production of appetite-regulating hormones, namely leptin and ghrelin [33]. It has been established that leptin and ghrelin play fundamentally opposite roles in the regulation of appetite and energy homeostasis. Leptin is a hormone secreted by white adipocytes, and its primary function is to regulate appetite by suppressing the desire to consume food, thereby stimulating energy expenditure. Ghrelin, on the other hand, is a hormone secreted by the stomach and regulates appetite by stimulating hunger sensations, thereby increasing fat production and promoting body growth [34]. Thus, it has been shown that after total sleep deprivation or

chronic sleep restriction in experimental participants, the level of ghrelin increases or the level of leptin decreases, which may contribute to weight gain and obesity in the long term [35]. Moreover, numerous studies have demonstrated that adolescents and young adults are significantly more prone to metabolic disorders and, consequently, obesity, compared to individuals over 40 years old. Several studies have reported an association between short sleep duration and the risk of obesity in children and adolescents, with estimates of an increased risk of up to 90% [36].

Effect on the immune system. Sleep is thought to be an important modulator of the immune response. For example, sleep deprivation can weaken the immune system and increase susceptibility to infection, so reduced sleep duration is also associated with an increased incidence of seasonal colds and flu. It has been shown that 7-8 hours of sleep can reduce the risk of catching a cold by about 20% [37]. Even minor sleep loss alters molecular processes that stimulate cellular immune activation and induce the production of inflammatory cytokines and markers of inflammation.

It has been shown that sleep deprivation leads to a decrease in natural killer cell activity, inhibition of interleukin IL-2 production (which increases NK and cytotoxic T cell activity and promotes B cell proliferation and immunoglobulin secretion) [38], and an increase in circulating markers of inflammation (such as interleukin IL-6, which is responsible for neutrophil production in the bone marrow, tumour necrosis factor TNFα, and immunoglobulin secretion), and an increase in the levels of circulating markers of inflammation (such as interleukin IL-6, which is responsible for neutrophil production in the bone marrow, tumour necrosis factor TNF-α, C-reactive protein, which acts as a marker of inflammatory processes or tissue damage, etc.) [39]. Thus, sleep deprivation induces functional changes in the pro-inflammatory cytokine response of monocytes.

Numerous studies of the normal sleep-wake cycle have shown that immune parameters such as the number of undifferentiated T cells and the production of anti-inflammatory cytokines peak during nighttime sleep (especially during NREMS), whereas the circulating number of immune cells with direct effector functions, such as cytotoxic natural killer cells, and the activity of anti-inflammatory cytokines peak during morning wakefulness [40]. In summary, blood levels of monocytes, T cells and NK cells (natural killer cells) follow a clear circadian rhythm related to the sleep-wake cycle.

Sleep deprivation has also been shown to reduce the antibody response to immunisation. An important feature of the immune system is its ability to 'remember' pathogens and respond more quickly when the same pathogen is encountered again. This system of immunological memory is also active during the production of antibodies following vaccination. This unique system of 'memory' and subsequent response consists of T lymphocytes, B lymphocytes and antibodies that circulate in the blood and other body fluids.

Sleep deprivation (<6 hours per night) during vaccination reduces the antibody response because the number of APCs (antigen-presenting cells) and T lymphocytes in circulation decreases during sleep and are redirected to the lymph nodes. This increases the likelihood that these two cell types will encounter each other, facilitating information transfer. In addition, as mentioned above, sleep promotes the production of pro-inflammatory cytokines, which further support the interaction between APCs and T cells[41].

Increased risk of cancer. Therefore, sleep disturbances can lead to immune suppression and a shift towards cytokine predominance, which in turn can lead to cellular mutations and significantly increase the risk of cancer. As mentioned earlier in this article, melatonin plays a crucial role in sleep regulation, but this hormone also has anti-inflammatory and, in particular, anti-tumour effects. It has been shown that the concentration of melatonin in blood serum is lower in individuals who habitually sleep for short periods (<6 hours/night) than in those who sleep for longer periods (>9 hours/night) [42]. Thus, sleep duration may influence melatonin levels, which determine the duration of light exposure and consequently the risk of developing cancer.

In experimental models of chemical carcinogenesis in rats, melatonin release has been shown to inhibit the initiation phase of the tumour process. One of the mechanisms by which this may occur is the ability of melatonin to inhibit the accumulation of DNA adducts (complexes formed by the binding of other chemicals to DNA) formed by carcinogens, which actually cause DNA damage and permanent changes (i.e. mutations and amplifications) leading to neoplastic transformation of cells and tissues[43]. Thus, as a result of disturbances in melatonin secretion during sleep, the risk of mutation in cells increases by about 35% in people who sleep less than 6-7 hours, have sleep disorders, or follow an incorrect daily routine.

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